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The Neurologic Aspects of Vertigo

Analysis of 400 Cases

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• **Of almost 8,000 patients referred for neurological consultation, 6.1 per cent had "dizziness" as a presenting complaint. Dizziness is a nonspecific complaint, used loosely to describe funny feelings in the head or lightheadedness by anxious or depressed patients; or it may mean vertigo—a hallucination of movement of self or surroundings in horizontal, rotatory or vertical direction. An analysis of 400 cases showed the complaint "dizziness" to be functional in about 25 per cent of patients. The cause in the remaining cases varied from epilepsy from cortical lesions, to lesion of the brain stem, such as tumors, vascular insufficiency, and multiple sclerosis, or to the peripheral neurone from Meniere's disease, and vestibular neuritis.**

Leading the patient out in a description of the kind of dizziness he feels may give clues that will help differentiate between true vertigo and functional disorder, particularly when considered against the information that is obtained in neurological examination.

THE ART OF MEDICINE, primarily concerned with clinical diagnosis, is of necessity preoccupied with symptoms of deranged function. The symptom *vertigo* demands attention not only because of its disruptiveness, but also because of its high clinical incidence. Thus, the 400 consecutive cases being here considered constituted an incidence of 6.1 per cent, as they were observed in the examination of almost 8,000 patients referred for neurological consultation. The series represents but a small proportion of all neurological cases in which vertigo is present; it consists of patients with a presenting complaint of dizziness or giddiness or a preliminary diagnosis of vertigo.

Of the galaxy of symptoms there is—with the exception of pain—probably no symptom personally experienced by so many physicians as vertigo—uncommonly from a pathological process, but commonly from unphysiological stimulation by motion while on the seas, in the air or on the merry-go-round, or by alcohol.

By derivation the term *vertigo* implies an hallucination of rotation of self or surroundings. In clinical neurology the term is used more comprehensively to include the descriptions of the sensations experienced by patients who have vertigo due to organic lesions of the vestibular apparatus resulting in dysequilibrium: An hallucination of movement of self or surroundings in horizontal, rotatory or vertical directions, varying in severity. Thus, a patient may be thrown to the ground on one occasion, while at another time have only a vague feeling of unsteadiness or uncertainty of movement, difficult to express

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in words. Some of these mildest forms may be dismissed as psychogenic, although readily accepted when seen as the end stage, lasting at times many days, of a severe attack of vertigo. The vertigo may be paroxysmal, chronic, postural or positional; or a single attack or several attacks may occur over many years.

Jongkees[9] said that vertigo was due to unphysiological stimulation of the vestibular organ. By experiments with subjects in a swing that was moved back and forth horizontally, he found that the direction of the acceleration given to the swing and of the acceleration of gravity form a parallelogram of accelerations with calculable direction; and normal subjects oscillated on the swing invariably confirmed this by their sensations. Thus, theoretically, if the subject is swinging head first, he will feel that he is tilting backwards, and, in fact, this is the sensation he does perceive. Vice versa, when the swing is moving feet first, he feels that he is tilting forward. It should be possible to place a normal person on the horizontal swing in such a position and subject him to such force, that the direction finds the "blind spot" of the labyrinth—that is, the position in which no otolith is in contact with its layer. This is found to be true in practice, the subject retaining no sensation at all of his position in space, despite intact proprioceptive and skin reflexes; and so, the movement is not perceived.

Vertigo is an impression in consciousness, not of actual movement, but an upset of the normal dynamic relationship existing between the body or the head and space, the vestibular mechanism being the proprioceptive organ for the head-neck positioning. As is well known, it has been demonstrated experimentally that this mechanism, along with the proprioceptors of the muscles, joints and spine, is reflexly capable of enabling the animal which has undergone a transection of the brain stem above the level of the red nuclei to adopt a normal posture and then, when displaced from that posture, to return to it by means of the righting reflexes. Physiological overstimulation of this mechanism, as occurs in acceleration or deceleration in certain planes or caloric stimulation, results in vertigo as do disease processes which disturb function. In the absence of such excessive stimuli or disorders, there is no impression in consciousness of the orientation of the body in space.

Vertigo may, however, occur when this orderly orientation is disturbed by an illusion of movement of space, as occurs when one is sitting in a stationary train and the nearby stationary train moves, or by an apparent disturbance of oneself with regard to space. As Hughlings Jackson[8] pointed out, the simplest form of vertigo is that which is produced by paralysis of an ocular muscle. This is primarily due to the faulty projection of the visual field of the affected eye, as a result of which the patient is presented with two visual spaces, the vestibular apparatus having the task of orientating the head in relation to each of its two proper but conflicting fields.

While the vestibular apparatus is sufficient to maintain posture unaided by vision or consciousness, both of these play important roles. Without vision one's extracorporeal space is very limited and righting reflexes can act unhampered. When the vestibular apparatus is damaged, vision may, and does, enable posture to be maintained; but with a normal vestibular mechanism, the wider the horizon and the greater the depth of space, the more difficult is the problem of orientation. Here, vision poses problems for the vestibular apparatus, rather than aids it. This is what appears to happen in the vertigo on a precipice or high platform. Does it play a part in psychogenic vertigo? Here, the vertigo characteristically occurs in the open. Meeting a person in a wide field may present a real problem with difficulty in maintaining equilibrium, as though vision was bringing into consciousness problems of illusion in spatial orientation to which the vestibular apparatus had to adjust.

PRESENT STUDY

Viewed against this background and after careful neurological and otological study, including audiometric and caloric examination, these 400 cases in the present series were considered to consist of:

Organic Vertigo	No. of Cases
Epilepsy	15
Posterior fossa lesions	52
Vestibular neuronitis	32
Miscellaneous peripheral causes	122
Traumatic	5
Undiagnosed	35
Total	261

Not Organic Vertigo	No. of Cases
Psychogenic	106
Epilepsy	29
Parkinson's disease	4
Total	139

CORTICAL VERTIGO

Vertigo and Epilepsy

Forty-four of the patients were diagnosed as having epilepsy. Twenty-nine of these, although referred with a diagnosis of vertigo, were found not to have organic vertigo. They had brief confusional episodes, partial loss of consciousness, or petit mal, while one had Stokes-Adams attacks from cardiac asystole. They had all described their attacks as

dizziness, but analysis of the history revealed the true condition, confirmed by neurological examination or by electroencephalography or response to anticonvulsive medication. In the case of the patient with Stokes-Adams syndrome, however, the condition was not diagnosed as such until he had an attack during a neurological examination, the asystole then being recognized.

Fifteen of these patients, however, had true vertigo, but again analysis revealed other aurae or subsequent loss of consciousness. Thus, a male aged 32 years complained of a hallucination of rotation of surroundings of two or three minutes' duration, at times preceded by paresthesiae in the fingers of the right hand and at times accompanied by vomiting. Loss of consciousness occasionally followed. On examination, he showed a mild aphasia, a partial right homonymous hemianopia and a mild right facial weakness. X-ray examination of the skull revealed a piece of shrapnel in the left posterior temporal area.

Symonds[13] expressed the opinion that vertigo was of no localizing value in cerebral tumors, and all the evidence supports this view in the presence of elevated intracranial pressure. Vertigo as an aura is not uncommon, occurring, in the experience of Gowers,[6] in 18 per cent of all cases of epilepsy. Exclusive of the present study, 50 cases of epilepsy with vertigo as an aura had features supportive of temporal lobe involvement—for example, commonly accepted temporal lobe aurae, focal electroencephalographic changes, dilatation of one temporal horn on pneumoencephalography, or even calcification observed roentgenographically.

Moreover, in one series of 100 cases of temporal lobe epilepsy, 31 of the patients had vertigo as an aura. Penfield,[12] during operations, was able to elicit vertigo by stimulation of the first temporal convolution. This suggests that an aura of vertigo (in the absence of raised intracranial pressure) usually indicates a discharge in the temporal lobe.

CENTRAL VERTIGO

Table 1 gives the incidence of posterior fossa lesions presenting with vertigo.

In 12 of the cases of vascular lesions, a diagnosis was arrived at only after one to four years' observation. The patients had attacks of vertigo varying in duration from 30 seconds to six or seven days with varying frequency, but without focal signs until the final, severe vertigo and vomiting immediately preceded a "stroke" with signs indicative of a brain stem lesion. In the other 12 cases these signs were present when the patients were first seen. The vertigo in these 24 patients was the presenting symptom in vertebro-basilar artery insufficiency or thrombosis.

TABLE 1.—*Incidence of Posterior Fossa Lesions Associated with Vertigo*

Posterior Fossa Lesions	No. of Cases
Brain stem vascular	24
Multiple sclerosis	16
Tumors	9
Hydrocephalus	1
Meningovascular lues	1
Olivo-ponto cerebellar syndrome	1
Vestibular neuronitis	32
Total	84

The tumors included one acoustic neuroma, two pontine gliomas, one brain stem astrocytoma and five cerebellar tumors. Only one, the brain stem astrocytoma, presented a diagnostic problem; in the other cases there were localizing signs and a progressive history. The patient with brain stem astrocytoma, a boy aged 16 years, first noticed that, on looking up when playing games, he invariably became violently dizzy—so much so that he would not assume that posture even for medical examination. The only neurological sign was a mild weakness of the right palate, but x-ray examination of the skull revealed calcification in the posterior fossa, proved at operation to be an astrocytoma. This case may be considered as an example of positional vertigo of the central type, first described by Lindsay.[10]

Edwards and Paterson[4] in their review of 157 cases of acoustic neuroma reported vertigo as the initial symptom in 18 per cent, but they concluded that when vertigo and deafness were not associated with nystagmus, with cerebellar incoordination or with impairment of corneal or facial sensation, it was unlikely to be due to an acoustic neuroma.

That vertigo may occur from a selective neuropathic effect on the vestibular neurone by streptomycin is well recognized. Dix and Hallpike,[3] when they defined a separate clinical entity, *vestibular neuronitis*, suggested that similar damage might occur from other toxic and unknown causes. Vestibular neuronitis is characterized by paroxysmal or even long-lasting (three to four weeks) vertigo with normal hearing on audiometric examination but a manifestation of canal paresis or paralysis on caloric testing. In a few of the patients who had this condition, the caloric abnormality later returned to normal. In several patients with vestibular neuronitis, the vertigo was the first and only manifestation of multiple sclerosis. In the present series, 16 cases of vertigo were due to multiple sclerosis, and in three of them vertigo was the first evidence of disease. The incidence of vertigo in acute multiple sclerosis is high and the sensation may be severe and prolonged. It has the same value in terms of

deciding on the multiplicity of lesions as the more commonly accepted symptom, diplopia.

Each of the five patients with traumatic vertigo had had a significant head injury with loss of consciousness, but without evidence of neurological lesions. In each, vertigo was of a positional type—that is, only with the head in a certain position in bed or at work did the vertigo occur. In two, an automobile mechanic and an engineer, it started only when they inspected automobiles or machinery from below, with the head tilted to the right. In each patient, nystagmus and vertigo occurred about five seconds after he was put in the critical position, then would disappear after 10 to 30 seconds (even though the position might be maintained). Dix and Hallpike[3] considered the lesion in such cases to be in the otolith. Nylén[11] expressed the opinion that when a particular position produces nystagmus and the direction of oscillation is not changed by changes of the position of the head, the lesion is peripheral; and when it is of a position-changing type, the direction of oscillation depending on the position of the head, the lesion is in the posterior fossa.

The four patients with Parkinson's disease in the present series were referred because of problems of dizziness, the extrapyramidal rigidity having gone unrecognized, as it often does when not suspected. None of them had true vertigo. On analysis, the problem was one of tending to lose balance in turns or on standing up or sitting down, and a feeling of insecurity when walking. It is of course possible for vertigo to occur when the underlying pathologic change affects the vestibular mechanism; but this was not the cause in these four cases.

PSYCHIC VERTIGO

The 106 patients with dizziness that was labelled "psychogenic" did not have true vertigo, although it is to be admitted that when vertigo is very mild, as it may be in a patient recovering from a brain stem lesion, the remaining complaint of a vague feeling of unsteadiness would be difficult to accept on its own as true vertigo. Moreover, as vertigo is very disturbing, it is not infrequent for patients to have a reaction of anxiety or depression, or even of hysterical elaboration, secondary to the organic vertigo.

In all of these patients, results of otologic, audiometric, caloric and neurologic examinations were within normal limits and there was evidence of anxiety, depression, hypochondriasis and other such emotional disturbances. In some the dizziness formed part of a mild injury syndrome with headache, and all showed pain and tenderness and some degree of spasm of the posterior cervical muscles. It may be suggested that tension receptors in these muscles, which form part of the head- and neck-righting mechanism, being affected by spasm, bombard the central vestibular mechanisms with afferent stimuli, and true vertigo results. Such a view is, however, not supported by the patient's description of the ensuing dizziness—a description which differs from the customary definition, being: "I fear I am going to fall"; "I feel pulled over to people when I am walking"; "I feel pulled over when I am out in the open and am afraid I am going to fall"; "I have a feeling of lightheadedness." In the majority, the problem was one of use of words—using "dizziness" to describe lightheadedness or a tight feeling in the head. In the others, there was a fear of open spaces or crowds, because of the ensuing sensation of dysequilibrium—a disturbance of the relationship of the individual to space, of an illusory character.

Although in the present series responses to caloric tests were normal, Hallpike and co-workers[7] reported that when caloric tests were quantitatively employed, in general neurotic patients were observed to have greater sensitivity than had other patients.

PERIPHERAL VERTIGO

Peripheral vertigo as observed in 122 patients was of three types:

1. *Spontaneous.* Eighty-two patients had spontaneous paroxysmal vertigo lasting from a few minutes to four hours, accompanied as a rule by nausea and vomiting, blurring of vision, tinnitus, distortion of hearing and exacerbation of tinnitus and deafness. Audiometry showed unilateral or bilateral perceptive deafness of varying degrees and loudness recruitment, while caloric responses showed some abnormality. Neurological signs were not present. Spontaneous paroxysmal vertigo of this type fitted the category of Meniere's disease, well described by Cawthorne[2] and many others.[14]

2. *Positional.* Twenty-six patients described vertigo in certain positions only—for example, with either the right or left ear down, the dizziness lasting only a few seconds and disappearing when the position was maintained. Rotatory nystagmus toward the involved ear was present for periods of from 5 to 30 seconds when the patient was placed in the critical position. Audiometry and caloric responses were normal. This disorder was self-limiting, lasting usually several months to a maximum of three years. Positional vertigo and nystagmus of this kind is well described by Dix and Hallpike.[3]

3. *From Destructive Lesions.* Fourteen patients had destructive lesions of the vestibular apparatus, most commonly otitis media with ample clinical evidence of local pathologic change.

Despite careful, comprehensive examination, no diagnosis was made in 35 patients although they gave a history of spontaneous vertigo of varying duration, at times recurring over months to five years. There is a tendency to attach labels, such as Meniere's syndrome, or even pseudo-Meniere's syndrome to such cases, but it appears preferable to resist attaching a label that might prevent an accurate diagnosis at a later date.

DIAGNOSIS

Encouraging the patient to describe the complaint of dizziness allows the first major separation of vertigo into the groups, organic and psychiatric. Some patients, particularly in the psychiatric group, resist such invitations, saying, "I am just dizzy"; but patience and the offering of alternative descriptions enable them to illustrate the complaint. This procedure helps in diagnosing epilepsy, whether or not accompanied by vertigo. It is only rarely that severe paroxysmal vertigo results in syncopal loss of consciousness, and when it does, consciousness is rapidly regained when the patient falls, even though vertigo may still persist.

Differentiation between central and peripheral vertigo is generally easy, but at times difficult. The presence of cochlear symptoms usually indicates a peripheral cause, except in cases of nerve lesions, as in acoustic neuroma. Observation of a minor neurological sign, such as absence of corneal reflex, or major signs of brain stem involvement settles the issue, as it does with many other neurological lesions. Pathological diagnosis is then established by further special neurological diagnostic procedures.

A familiarity with the natural history of Meniere's disease and positional vertigo usually makes recognition fairly easy; but it may be necessary to employ both otologic and neurologic diagnostic procedures to come to a definitive diagnosis, or to recognize that, although the vertigo appears organic, the cause is not ascertainable.

The age of onset is of little value, although, speaking in general terms, in the present series vertigo occurred in vascular diseases chiefly in the fifth and sixth decades, in Meniere's disease in the third, fourth and fifth decades, in vestibular neuronitis and multiple sclerosis in the second and third decades.

Except that positional vertigo appeared most frequently in females, there was no essential difference in sex incidence.

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REFERENCES

1. Alpers, B. J.: Vertigo and Dizziness, Grune and Stratton, New York, 1958.

2. Cawthorne, T.: Meniere's disease, Ann. Otol. Rhin. & Laryng., 56:18-38, March 1947.

3. Dix, M. R., and Hallpike, C. S.: The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system, Proc. Roy. Soc. Med., 45:341-354, 1952.

4. Edwards, C. H., and Paterson, J. H.: A review of the symptoms and signs of acoustic neurofibromata, Brain, 74:144-190, 1951.

5. Fletcher, R.: Postural vertigo and positional nystagmus, Calif. Med., 77:377-379, 1952.

6. Gowers, W. R.: Epilepsy and Other Chronic Convulsive Diseases, J. A. Churchill, London, 1901.

7. Hallpike, C. S., Harrison, M. S., and Slater, E.: Abnormalities of caloric test results in certain varieties of mental disorder, Acta Oto-laryng., 39:151-159, June 1951.

8. Jackson, J. H.: Selected Writings of John Hughlings Jackson, edited by James Taylor, Hodder and Stoughton, London, 1931.

9. Jongkees, L. B. W.: Some remarks on vestibular examination, Proc. Roy. Soc. Med., 45:127-133, 1952.

10. Lindsay, J. R.: Postural vertigo and positional nystagmus, Ann. Otol. Rhin. & Laryng., 60:1134-1149, Dec. 1951.

11. Nylén, C. O.: Positional nystagmus; a review and future prospects, J. Laryng. & Otol., 64:295-318, 1950.

12. Penfield, W., and Gage, L.: Cerebral localization of epileptic manifestations, Arch. Neurol. & Psychiat., 30:709-727, Oct. 1933.

13. Symonds, Sir C.: The significance of vertigo in neurological diagnosis, J. Laryng. & Otol., 66:295-301, July 1952.

14. Symonds, Sir C.: Section of Otology, Proc. Roy. Soc. Med., 45:354, Jan. 1952.